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FILE 'BIOSIS, CAPLUS, EMBASE, MEDLINE' ENTERED AT 14:41:19 ON 27 JAN 2007
L1 117 S ADIPOCYTOKINES AND PD<=20020726
L2 505 S ADIPONECTIN AND PD<=20020726
L3 23383 S LEPTIN AND PD<=20020726
L4 466840 S ("TYPE 2 DIABETES") OR OBESITY OR ("CARDIOVASCULAR DISEASE")
L5 5288 S ("WHITE ADIPOSE TISSUE") AND PD<=20020726
L6 0 S ("GLOBULAR DOMAIN" (XW) ADIPONECTIN) AND PD<=20020726
L7 56 S L1 AND L2
L8 46 DUP REM L7 (10 DUPLICATES REMOVED)
L9 161 S L2 AND L3
L10 92 DUP REM L9 (69 DUPLICATES REMOVED)
L11 60 S L10 AND L4
L12 60 DUP REM L11 (0 DUPLICATES REMOVED)
L13 15 S L10 AND L5
L14 15 DUP REM L13 (0 DUPLICATES REMOVED)
L15 328 S L2 AND L4
L16 191 DUP REM L15 (137 DUPLICATES REMOVED)

=> D BIB ABS L12 4-25

L12 ANSWER 4 OF 60 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2002:775209 CAPLUS <<LOGINID::20070127>>
DN 138:37209
TI Removal of visceral fat prevents insulin resistance and glucose intolerance of aging: an adipokine-mediated process?
AU Gabriely, Ilan; Ma, Xiao Hui; Yang, Xiao Man; Atzmon, Gil; Rajala, Michael W.; Berg, Anders H.; Scherer, Phillip; Rossetti, Luciano; Barzilai, Nir
CS Diabetes Research and Training Center and Division of Endocrinology, Department of Medicine, Institute for Aging Research, Albert Einstein College of Medicine, Bronx, NY, 10461, USA
SO Diabetes (2002), 51(10), 2951-2958
CODEN: DIAEAZ; ISSN: 0012-1797
PB American Diabetes Association
DT Journal
LA English
AB Age-dependent changes in insulin action and body fat distribution are risk factors for the development of type 2 diabetes . To examine whether the accumulation of visceral fat (VF) could play a direct role in the pathophysiol. of insulin resistance and type 2 diabetes, we monitored insulin action, glucose tolerance, and the expression of adipo-derived peptides after surgical removal of VF in aging (20-mo-old) F344/Brown Norway (FBN) and in Zucker Diabetic Fatty (ZDF) rats. As expected, peripheral and hepatic insulin action were markedly impaired in aging FBN rats, and extraction of VF (accounting for .apprx.18% of their total body fat) was sufficient to restore peripheral and hepatic insulin action to the levels of young rats. When examined at the mechanistic level, removal of VF in ZDF rats prevented the progressive decrease in insulin action and delayed the onset of diabetes, but VF extraction did not alter plasma free fatty acid levels. However, the expression of tumor necrosis factor-a and leptin in s.c. (SC) adipose tissue were markedly decreased after VF removal (by approx. three- and twofold, resp.). Finally, extracted VF retained .apprx.15-fold higher resistin mRNA compared with SC fat. Our data suggest that insulin resistance and the development of diabetes can be significantly reduced in aging rats by preventing the age-dependent accumulation of VF. This study documents a cause-and-effect relationship between VF and major components of the metabolic syndrome.
RE.CNT 39 THERE ARE 39 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L12 ANSWER 5 OF 60 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2002:824519 CAPLUS <<LOGINID::20070127>>
DN 137:335596
TI Significance of adipocytokine, fat-derived hormones, in metabolic syndrome
AU Shimomura, Iichiro; Funahashi, Tohru; Matsuzawa, Yuji

CS Grad. Sch. Biofunct. Res., Osaka Univ., Japan
SO Tanpakushitsu Kakusan Koso (2002), 47(14), 1896-1903
CODEN: TAKKAJ; ISSN: 0039-9450
PB Kyoritsu Shuppan
DT Journal; General Review
LA Japanese
AB A review on the pathophysiol. roles and clin. significance of adipocytokines, adipocyte-derived hormones, in obesity-caused metabolic syndromes including diabetes mellitus, hyperlipidemia, and atherosclerosis, focusing on PAI-1, TNF-a, leptin, and adiponectin.

L12 ANSWER 6 OF 60 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2002:768894 CAPLUS <<LOGINID::20070127>>
DN 138:167458
TI Syndrome of insulin resistance. Adipocytokines
AU Kishida, Ken; Funahashi, Tohru
CS Dep. Internal Med. Molecular Sci., Grad. Sch. Med., Osaka Univ., Japan
SO Saishin Igaku (2002), 57(8), 1799-1805
CODEN: SAIGAK; ISSN: 0370-8241
PB Saishin Igakusha
DT Journal; General Review
LA Japanese
AB A review, on the roles of adipocytokines (fatty acids, glycerol, TNFa, leptin, adiponectin) in the pathogenesis of insulin resistance (IR) and IR-associated syndromes (obesity, type 2 diabetes, hypertension, lipid metabolic disorders, hyperinsulinemia, atherosclerosis).

L12 ANSWER 7 OF 60 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2002:746437 CAPLUS <<LOGINID::20070127>>
DN 138:150908
TI Central role of adipocytokine on metabolic syndrome
AU Shimomura, Iichiro; Funahashi, Tohru; Kihara, Shinji; Matsuzawa, Yuji
CS Dep. of Frontier Bioscience, Graduate School of Frontier Bioscience, Osaka University, Japan
SO Jikken Igaku (2002), 20(12), 1762-1767
CODEN: JIIGEF; ISSN: 0288-5514
PB Yodosha
DT Journal; General Review
LA Japanese
AB A review, on the roles of adipocytokines (PAI-I, TNF-a, leptin, and adiponectin) on metabolic syndromes, such as obesity, diabetes, hyperlipemia, and atherosclerosis.

L12 ANSWER 8 OF 60 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2002:469099 CAPLUS <<LOGINID::20070127>>
DN 137:183818
TI Gene expression profile of rat adipose tissue at the onset of high-fat-diet obesity
AU Li, Jinping; Yu, Xinxin; Pan, Wentong; Unger, Roger H.
CS Gifford Laboratories, Touchstone Center for Diabetes Research, Department of Internal Medicine, University of Texas Southwestern Medical Center, Dallas, TX, 75390-8854, USA
SO American Journal of Physiology (2002), 282(6, Pt. 1), E1334-E1341
CODEN: AJPHAP; ISSN: 0002-9513
PB American Physiological Society
DT Journal
LA English
AB Morbid obesity is the result of massive expansion of white adipose tissue (WAT) and requires recruitment of adipocyte precursor cells and their supporting infrastructure. To characterize the change in the expression profile of the preexisting WAT at the start of obesity , when adipocyte hypertrophy is present but hyperplasia is still minimal, the authors employed a cDNA subtraction screen for genes differentially expressed in epididymal fat pads harvested 1 wk after the start of a 60% fat diet. Ninety-six genes were upregulated by at least 50% above the WAT of control rats receiving a 4% fat diet. Of these genes, 30 had not previously been identified. Sixteen of the 96 genes, including leptin, adipocyte complement-related protein 30 kDa, and resistin, were predicted to encode a signal peptide. Ten of the 16 had been previously identified in other tissues and implicated in cell growth, proliferation, differentiation, cell cycle control, and angiogenesis. One was a novel gene. Twenty-nine novel fragments were identified. Thus, at the onset of high-fat-diet-induced obesity in rats, adipose tissue increases its expression of factors previously implicated in the expansion of non-adipocyte tissues and of several uncharacterized novel factors. The only one of these thus far characterized functionally was found to promote lipogenesis.
RE.CNT 40 THERE ARE 40 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L12 ANSWER 9 OF 60 MEDLINE on STN
AN 2002698566 MEDLINE <<LOGINID::20070127>>
DN PubMed ID: 12388167
TI Adiponectin is stimulated by adrenalectomy in ob/ob mice and is highly correlated with resistin mRNA.
AU Makimura Hideo; Mizuno Tooru M; Bergen Hugo; Mobbs Charles V
CS Neurobiology of Aging Laboratories, Fishberg Center for Neurobiology and Department of Geriatrics and Adult Development, Mount Sinai School of

Medicine, New York, New York 10029,, USA.
SO American journal of physiology. Endocrinology and metabolism, (2002 Dec) Vol. 283, No. 6, pp. E1266-71. Electronic Publication: 2002-08-13.
Journal code: 100901226. ISSN: 0193-1849.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200212
ED Entered STN: 17 Dec 2002
Last Updated on STN: 5 Jan 2003
Entered Medline: 9 Dec 2002
AB Plasma levels of the adipocyte product adiponectin, a putative insulin-sensitizing agent, are reduced in obesity, whereas plasma levels of resistin, an agent that some believe to confer insulin resistance, are thought to increase with obesity. Because adrenalectomy can increase insulin sensitivity, we hypothesized that adrenalectomy would increase expression of adiponectin and decrease expression of resistin. Therefore, we measured adiponectin mRNA, adiponectin peptide, and resistin mRNA in adrenalectomized ob/ob mice. Adrenalectomy restored adiponectin expression in ob/ob mice to wild-type levels and stimulated adiponectin peptide to above wild-type levels. Surprisingly, expression of adiponectin and resistin was highly positively correlated even after statistical removal of effects of insulin, glucose, and adiposity. In addition, adiponectin and resistin expression were also highly correlated in diet-induced obese mice. The data support a role for adiponectin in mediating some effects of adrenalectomy on insulin sensitivity.

L12 ANSWER 10 OF 60 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2002:949636 CAPLUS <<LOGINID::20070127>>
DN 138:220160
TI Resistin and adiponectin expression in visceral fat of obese rats: effect of weight loss
AU Milan, Gabriella; Granzotto, Marnie; Scarda, Alessandro; Calcagno, Alessandra; Pagano, Claudio; Federspil, Giovanni; Vettor, Roberto
CS Endocrine-Metabolic Laboratory, Internal Medicine, Department of Medical and Surgical Sciences, University of Padova, Padua, 35128, Italy
SO Obesity Research (2002), 10(11), 1095-1103
CODEN: OBREFR; ISSN: 1071-7323
PB North American Association for the Study of Obesity
DT Journal
LA English
AB Obesity-related insulin resistance is closely associated with visceral fat accumulation. Several adipocyte-secreted mols. have been implicated in the development of type 2 diabetes, among them, the recently discovered adiponectin and resistin proteins. Some of these adipocytokines are also present in the immune system, thus suggesting an intriguing functional connection. We determined adiponectin and resistin expressions in visceral (VAT) and s.c. adipose tissue of lean and obese Zucker (fa/fa) rats using reverse-transcription polymerase chain reaction. Moreover, we analyzed the variations after body-weight reduction in food-restricted obese rats. Resistin and adiponectin expression was significantly lower in VAT of genetically obese in comparison with lean rats; no differences were observed when s.c. adipose tissues of the same animals were compared. Weight loss resulted in an increase of adiponectin expression in VAT, whereas a further significant decrease in resistin mRNA level was observed Resistin is also present and equally expressed in splenocytes of lean and obese rats. Adiponectin and resistin are down-regulated in VAT of obese rats. Adiponectin expression is restored to normal levels after body-weight reduction, supporting its link with obesity -related insulin resistance. On the contrary, the further decrease of resistin mRNA after weight loss does not support the hypothesis that resistin may play a causative role in insulin resistance in obese rats. Moreover, we demonstrated the presence of resistin in immunocompetent cells in both humans and rats, thus adding another factor to the list of mols. that adipose tissue shares with the immune system.
RE.CNT 47 THERE ARE 47 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L12 ANSWER 11 OF 60 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN
AN 2002449010 EMBASE <<LOGINID::20070127>>
TI Cardiovascular risks in obesity.
AU Uchegbu E.C.; Kopelman P.G.
CS Dr. E.C. Uchegbu, Dept. of Diabetes and Metabolism, 5th Floor Alexandra Wing, Royal London Hospital, Turner Street, London E1 1BB, United Kingdom. e.c.uchegbu@qmul.ac.uk
SO Journal of Endocrinological Investigation, (2002) Vol. 25, No. 10, pp. 915-918. .
Refs: 38
ISSN: 0391-4097 CODEN: JEIND7
CY Italy
DT Journal; General Review
FS 029 Clinical Biochemistry
018 Cardiovascular Diseases and Cardiovascular Surgery
005 General Pathology and Pathological Anatomy
003 Endocrinology

017 Public Health, Social Medicine and Epidemiology
LA English
ED Entered STN: 3 Jan 2003
Last Updated on STN: 3 Jan 2003
DATA NOT AVAILABLE FOR THIS ACCESSION NUMBER

L12 ANSWER 12 OF 60 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2003:65783 CAPLUS <<LOGINID::20070127>>
DN 138:265785
TI Adipose tissue hormones
AU Guerre-Millo, M.
CS Centre de Recherche des Cordeliers, Universite Pierre et Marie Curie, Paris, 75006, Fr.
SO Journal of Endocrinological Investigation (2002), 25(10), 855-861
CODEN: JEIND7; ISSN: 0391-4097
PB Editrice Kurtis s.r.l.
DT Journal; General Review
LA English
AB A review. It is now widely accepted that white adipose tissue (WAT) secretes a number of peptide hormones, including leptin, several cytokines, adipsin and acylation-stimulating protein (ASP), angiotensinogen, plasminogen activator inhibitor-1 (PAI-1), adiponectin, resistin etc., and also produces steroids hormones. This newly discovered secretory function has shifted the authors' view of WAT, which is no longer considered only an energy storage tissue but a major endocrine organ, at the heart of a complex network influencing energy homeostasis, glucose and lipid metabolism, vascular homeostasis, immune response and even reproduction Virtually all known adipose secreted proteins are dysregulated when the WAT mass is markedly altered, either increased in the obese state or decreased in lipoatrophy. This strongly implicates adiposesecreted products in the ethiopathol. and/or complications of both obesity and cachexia. This review discusses the physiol. relevance of adipose secretion by focusing on protein and steroid hormones. Regulation of WAT secretion by the major regulatory factors impinging on the adipocytes, i.e., insulin, glucocorticoids, catecholamines and thiazolidinediones (TZD) will be addressed. The rationale for therapeutic strategies aimed at compensating adverse effects resulting from overprodn. or lack of a specific adipose secretory product will be discussed.
RE.CNT 53 THERE ARE 53 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L12 ANSWER 13 OF 60 MEDLINE on STN
AN 2002476461 MEDLINE <<LOGINID::20070127>>
DN PubMed ID: 12238130
TI Glucose intolerance in visceral fat syndrome.
AU Matsuzawa Yuji
CS Department of Internal Medicine and Molecular Science, Osaka University Graduate School.
SO Nippon rinsho. Japanese journal of clinical medicine, (2002 Jul) Vol. 60 Suppl 7, pp. 746-51. Ref: 14
Journal code: 0420546. ISSN: 0047-1852.
CY Japan
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
LA Japanese
FS Priority Journals
EM 200211
ED Entered STN: 20 Sep 2002
Last Updated on STN: 13 Dec 2002
Entered Medline: 20 Nov 2002

L12 ANSWER 14 OF 60 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2002:328227 CAPLUS <<LOGINID::20070127>>
DN 137:335603
TI Obesity: Molecular mechanism of obesity and its complications
AU Shimomura, Iichiro; Funahashi, Tohru; Matsuzawa, Yuji
CS Department of Internal Medicine and Molecular Science, Graduate School of Medicine, Osaka University, Japan
SO Saishin Igaku (2002), 57(March, Zokango, Seikatsu Shukanbyo, Zenpen), 708-717
CODEN: SAIGAK; ISSN: 0370-8241
PB Saishin Igakusha
DT Journal; General Review
LA Japanese
AB A review on mol. factors (especially PAI-1, TNF-a, leptin, adiponectin, and resistin) that are related to lipid metabolism and visceral fat accumulation in human.

L12 ANSWER 15 OF 60 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN
AN 2002422400 EMBASE <<LOGINID::20070127>>
TI Adiponectin: A link between excess adiposity and associated comorbidities?.
AU Ukkola O.; Santaniemi M.
CS O. Ukkola, Department of Internal Medicine, Biocenter Oulu, University of Oulu, Kajaanintie 50, 90220 Oulu, Finland. olavi.ukkola@oulu.fi
SO Journal of Molecular Medicine, (2002) Vol. 80, No. 11, pp. 696-702. .

Refs: 69
ISSN: 0946-2716 CODEN: JMLME8
CY Germany
DT Journal; General Review
FS 003 Endocrinology
006 Internal Medicine
030 Pharmacology
037 Drug Literature Index
LA English
SL English
ED Entered STN: 5 Dec 2002
Last Updated on STN: 5 Dec 2002
AB Adiponectin is a novel polypeptide that is highly specific to adipose tissue. In contrast to other adipocy-tokines, adiponectin levels are decreased in obesity and associated comorbidities, such as type 2 diabetes. Decreased expression of adiponectin is correlated with insulin resistance. It has been suggested that several agents, such as tumor necrosis factor a, could mediate their effects on insulin metabolism through modulating adiponectin secretion from adipocytes. The mechanisms for the development of atherosclerotic vascular disease in obese individuals are largely unknown. Several findings support the interesting hypothesis that adiponectin could be a link between obesity and related atherosclerosis. First, adiponectin levels are lower in patients with coronary artery disease. Second, adiponectin modulates endothelial function and has an inhibitory effect on vascular smooth muscle cell proliferation. Moreover, adiponectin is accumulated more preferably to the injured vascular wall than intact vessels and has been shown to suppress macrophage-to-foam cell transformation. Adiponectin may also be involved in the modulation of inflammation. Thiazolidinediones, antiatherogenic and other effects have been explained by their direct enhancing effect on adiponectin. In conclusion, adiponectin has anti-inflammatory and anti-atherogeneic effects as well as multiple beneficial effects on metabolism. Therefore it is not a surprise that adiponectin therapy has been tested in animal models of obesity, and it has been shown to ameliorate hyperglycemia and hyperinsulinemia without inducing weight gain or even inducing weight loss in some studies. Unlike agents that exert their effects centrally, adiponectin's effects seem to be peripherally mediated. The evidence of an association between adiponectin and the metabolic and cardiovascular complications of obesity is growing all the time.

L12 ANSWER 16 OF 60 MEDLINE on STN
AN 2002670544 MEDLINE <<LOGINID::20070127>>
DN PubMed ID: 12430302
TI Glucose metabolism in adipose tissue.
AU Inoue Atsushi; Tobe Kazuyuki; Suzuki Ryo; Kadowaki Takashi
CS Department of Internal Medicine, Graduate School of Medicine, University of Tokyo.
SO Nippon rinsho. Japanese journal of clinical medicine, (2002 Oct) Vol. 60 Suppl 10, pp. 673-80. Ref: 21
Journal code: 0420546. ISSN: 0047-1852.
CY Japan
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
LA Japanese
FS Priority Journals
EM 200302
ED Entered STN: 15 Nov 2002
Last Updated on STN: 21 Feb 2003
Entered Medline: 20 Feb 2003

L12 ANSWER 17 OF 60 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2003:262668 CAPLUS <<LOGINID::20070127>>
DN 138:399708
TI Relationship between IL-6, leptin and adiponectin and variables of fibrinolysis in overweight and obese hypertensive patients
AU Skurk, T.; van Harmelen, V.; Lee, Y. -M.; Wirth, A.; Hauner, H.
CS German Diabetes Research Institute at the Heinrich-Heine-University, Dusseldorf, 40225, Germany
SO Hormone and Metabolic Research (2002), 34(11/12), 659-663
CODEN: HMMRA2; ISSN: 0018-5043
PB Georg Thieme Verlag
DT Journal
LA English
AB Impaired fibrinolysis is a common finding in obese humans. This condition is now considered as an established risk factor for thromboembolic complications. Furthermore, obesity is characterized by a specific pattern of circulating concns. of fat-cell products interleukin-6 (IL-6), leptin, and adiponectin. The aim of our study was to investigate the relationship between these proteins and selected variables of the fibrinolytic system in 74 mildly hypertensive, overweight subjects. Circulating IL-6 and leptin levels showed a pos. association with BMI ($r = 0.24$, $p = 0.04$ and $r = 0.70$, $p < 0.0001$), whereas adiponectin was not correlated to BMI. Interestingly, IL-6 was also pos. associated with t-PA/PAI-1 complexes after adjustment for BMI and other anthropometric variables. Leptin was pos. correlated with PAI-1 activity and antigen ($r = 0.32$, $p = 0.006$ and $r = 0.37$, $p < 0.001$, resp.) and neg. with t-PA activity ($r = -0.27$, $p = 0.03$). However, these assocns. lost significance after correction for BMI or HOMA, an insulin

sensitivity index. In contrast, adiponectin levels were independently and neg. correlated with PAI-1 antigen (r = -0.26, p = 0.04, after correction for BMI). In conclusion, our study provides further evidence that IL-6, leptin, and adiponectin are associated with impaired fibrinolysis in overweight hypertensive humans.
RE.CNT 27 THERE ARE 27 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L12 ANSWER 18 OF 60 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN
AN 2005364778 EMBASE <<LOGINID::20070127>>
TI Adipose tissue as an endocrine organ.
AU Prins J.B.
CS Dr. J.B. Prins, Princess Alexandra Hospital, Ipswich Rd, Woolloongabba, QLD 4102, Australia
SO Best Practice and Research in Clinical Endocrinology and Metabolism, (2002) Vol. 16, No. 4, pp. 639-651. .
Refs: 97
ISSN: 1521-690X CODEN: BPRCE
CY United Kingdom
DT Journal; General Review
FS 003 Endocrinology
029 Clinical Biochemistry
LA English
SL English
ED Entered STN: 27 Oct 2005
Last Updated on STN: 27 Oct 2005
AB Adipose tissue is a highly active endocrine organ secreting a range of soluble products with both local and distant actions. These hormones have important roles in metabolism, reproduction, cardiovascular function and immunity. It is now evident that adipose endocrine function directly influences other organ systems, including the brain, liver and skeletal muscle. The endocrine function of adipose tissue is significantly regulated by nutritional status, and both are inextricably linked to the energy storage role of adipose tissue. This chapter highlights the endocrinology of adipose tissue by concentrating on functional aspects of the secreted products. The data of particular relevance to humans are highlighted, and areas in need of future research are suggested. .COPYRGT. 2002 Elsevier Science Ltd. All rights reserved.

L12 ANSWER 19 OF 60 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2003:262661 CAPLUS <<LOGINID::20070127>>
DN 138:396443
TI Differential gene expression between visceral and subcutaneous fat depots
AU Atzmon, G.; Yang, X. M.; Muzumdar, R.; Ma, X. H.; Gabriely, I.; Barzilai, N.
CS Institute for Aging Research & Diabetes Research and Training Center Department of Medicine, Albert Einstein College of Medicine, Bronx, NY, 10461, USA
SO Hormone and Metabolic Research (2002), 34(11/12), 622-628
CODEN: HMMRA2; ISSN: 0018-5043
PB Georg Thieme Verlag
DT Journal
LA English
AB Abdominal obesity has been linked to the development of insulin resistance and Type 2 diabetes mellitus (DM2). By surgical removal of visceral fat (VF) in a variety of rodent models, we prevented insulin resistance and glucose intolerance, establishing a cause-effect relationship between VF and the metabolic syndrome. To characterize the biol. differences between visceral and peripheral fat depots, we obtained perirenal visceral (VF) and s.c. (SC) fat from 5 young rats. We extracted mRNA from the fat tissue and performed gene array hybridization using Affymetrix technol. with a platform containing 9000 genes. Out of the 1660 genes that were expressed in fat tissue, 297 (17.9%) genes show a two-fold or higher difference in their expression between the two tissues. We present the 20 genes whose expression is higher in VF fat (by 3-7 fold) and the 20 genes whose expression is higher in SC fat (by 3-150 fold), many of which are predominantly involved in glucose homeostasis, insulin action, and lipid metabolism We confirmed the findings of gene array expression and quantified the changes in expression in VF of genes involved in insulin resistance (PPARg leptin) and its syndrome (angiotensinogen and plasminogen activating inhibitor-1, PAI-1) by real-time PCR (qRT-PCR) technol. Finally, we demonstrated increased expression of resistin in VF by around 12-fold and adiponectin by around 4-fold, peptides that were not part of the gene expression platform. These results indicate that visceral fat and s.c. fat are biol. distinct.
RE.CNT 29 THERE ARE 29 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L12 ANSWER 20 OF 60 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
AN 2002:387248 BIOSIS <<LOGINID::20070127>>
DN PREV200200387248
TI Resistin and adiponectin expression in lean and obese Zucker rats.
AU Blaylock, Matthew L. [Reprint author]; Nagy, Tim R. [Reprint author]
CS Nutrition Sciences, University of Alabama at Birmingham, 1675 University Blvd, Birmingham, AL, 35294, USA

SO FASEB Journal, (March 20, 2002) Vol. 16, No. 4, pp. A603. print.
Meeting Info.: Annual Meeting of the Professional Research Scientists on Experimental Biology. New Orleans, Louisiana, USA. April 20-24, 2002.
CODEN: FAJOEC. ISSN: 0892-6638.
DT Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LA English
ED Entered STN: 17 Jul 2002
Last Updated on STN: 17 Jul 2002
AB The mechanisms underlying obesity and type 2 diabetes remain to be elucidated. Two novel adipose-derived cytokines, resistin and adiponectin, have been implicated in these processes. The purpose of this study was to determine the expression of resistin and adiponectin in lean and fatty Zucker rats over a range of ages. Animals (n=9-10/group) were euthanized at 6, 7, 10, and 14 weeks of age and epididymal white adipose tissue was collected. The results showed that the fatty rats weighed significantly more, had greater adipose tissue mass as well as higher levels of plasma leptin, insulin, free-fatty acids, and triglycerides ($p<0.05$). Within each age class, the expression of resistin and adiponectin was reduced in the fatty compared to the lean Zucker rats ($p<0.05$). Our results are in agreement with recently published data suggesting that the expression of resistin and adiponectin is reduced with obesity and increasing insulin resistance.

L12 ANSWER 21 OF 60 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2002:424393 CAPLUS <<LOGINID::20070127>>
DN 138:2729
TI A novel transgenic mouse model of visceral fat obesity and metabolic syndrome
AU Masuzaki, Hiroaki; Flier, Jeffrey S.
CS Division of Endocrinology, Diabetes and Metabolism, Department of Medicine, Harvard Medical School and Beth Israel Deaconess Medical Center, Boston, MA, USA
SO Molecular Medicine (Tokyo, Japan) (2002), 39(4), 464-474
CODEN: MOLMEL; ISSN: 0918-6557
PB Nakayama Shoten
DT Journal; General Review
LA Japanese
AB A review. The topics discussed are (1) visceral fat obesity and metabolic disorders; (2) transgenic mouse (aP2 HSD-1 mouse) model of visceral fat obesity by overexpressing adipose tissue specific 11b-hydroxysteroid dehydrogenase type 1 (11b-HDS-1); (3) decreased energy metabolism, glucose tolerance, and insulin sensitivity in aP2 HSD-1 mice; (4) leptin resistance, visceral obesity, increased expression of lipoprotein lipase, angiotensinogen, and tumor necrosis factor-a, and decreased expression of adiponectin and resistin in adipose tissues of aP2 HSD-1 mice; and (5) increases in free fatty acids, corticosterone, phosphoenolypyruvate carboxykinase (PEPCK), and glucose-6-phosphatase (G6Pase) in liver of aP2 HSD-1 mice.

L12 ANSWER 22 OF 60 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN
AN 2003447834 EMBASE <<LOGINID::20070127>>
TI Slimming down without DGAT.
AU Brazil M.
SO Nature Reviews Drug Discovery, (2002) Vol. 1, No. 6, pp. 408. .
Refs: 1
ISSN: 1474-1776 CODEN: NRDDAG
CY United Kingdom
DT Journal; Note
FS 003 Endocrinology
029 Clinical Biochemistry
030 Pharmacology
037 Drug Literature Index
LA English
ED Entered STN: 20 Nov 2003
Last Updated on STN: 20 Nov 2003
DATA NOT AVAILABLE FOR THIS ACCESSION NUMBER

L12 ANSWER 23 OF 60 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2003:167589 CAPLUS <<LOGINID::20070127>>
DN 138:151453
TI The morbid condition caused by insulin resistance in the obese patient with diabetes. Homology of leptin, adiponectin and LPL mass
AU Takahashi, Toshikazu; Mochihara, Yuki; Kodate, Shinya; Mashimo, Ikuo; Tazawa, Hiromitsu; Taira, Yoshihisa; Yano, Masao; Shimomura, Koji; Maehata, Eisuke; Shiba, Teruo; Yamakado, Minoru; Inoue, Minoru; Taniyama, Matsuo; Suzuki, Seiji
CS Quality Assurance Office, Sumikin Bioscience K. K., Sagamihara, 229-1125, Japan
SO Seibutsu Shiryo Bunseki (2002), 25(5), 385-391
CODEN: SSBUEL; ISSN: 0913-3763
PB Seibutsu Shiryo Bunseki Kagakkai
DT Journal
LA Japanese
AB We compared the decrease in insulin resistivity in obese patients with diabetes using the insulin resistance index (HOMA-R) and the physiol. active substances secreted by adipose tissue (LPL mass, leptin, adiponectin). Changes in the homologous levels were investigated at the same time. As the HOMA-R progressed, leptin became

kinetic and adiponectin increased, showing an antagonistic relationship. As for the adipocytokine correlation, in the group with a BMI exceeding 25 kg/m2, correlations of BMI vs. leptin ($r = 0.694$, $p = 0.0003$) and adiponectin vs. LPL mass ($r = 0.618$, $p < 0.0022$) were recognized, and the same when compared with the group in which HOMA-R was over 2.0. From these results, insulin resistivity showed a characteristic morbid condition.

L12 ANSWER 24 OF 60 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2002:587011 CAPLUS <<LOGINID::20070127>>
DN 137:382874
TI Increased fat intake, impaired fat oxidation, and failure of fat cell proliferation result in ectopic fat storage, insulin resistance, and type 2 diabetes mellitus
AU Ravussin, Eric; Smith, Steven R.
CS Pennington Biomedical Research Center, Baton Rouge, LA, 70808-4124, USA
SO Annals of the New York Academy of Sciences (2002), 967(Lipids and Insulin Resistance), 363-378
CODEN: ANYAA9; ISSN: 0077-8923
PB New York Academy of Sciences
DT Journal; General Review
LA English
AB A review. It is widely accepted that increasing adiposity is associated with insulin resistance and increased risk of type 2 diabetes. The predominant paradigm used to explain this link is the portal/visceral hypothesis. This hypothesis proposes that increased adiposity, particularly in the visceral depots, leads to increased free fatty acid flux and inhibition of insulin action via Randle's effect in insulin-sensitive tissues. Recent data do not entirely support this hypothesis. As such, two new paradigms have emerged that may explain the established links between adiposity and disease. Three lines of evidence support the ectopic fat storage syndrome. First, failure to develop adequate adipose tissue mass in either mice or humans, also known as lipodystrophy, results in severe insulin resistance and diabetes. This is thought to be the result of ectopic storage of lipid into liver, skeletal muscle, and the pancreatic insulin-secreting beta cell. Second, most obese patients also shunt lipid into the skeletal muscle, the liver, and probably the beta cell. The importance of this finding is exemplified by several studies demonstrating that the degree of lipid infiltration into skeletal muscle and liver correlates highly with insulin resistance. Third, increased fat cell size is highly associated with insulin resistance and the development of diabetes. Increased fat cell size may represent the failure of the adipose tissue mass to expand and thus to accommodate an increased energy influx. Taken together, these three observations support the acquired lipodystrophy hypothesis as a link between adiposity and insulin resistance. The endocrine paradigm developed in parallel with the ectopic fat storage syndrome hypothesis. Adipose tissue secretes a variety of endocrine hormones, such as leptin, interleukin-6, angiotensin II, adiponectin (also called ACRP30 and adipoQ), and resistin. From this viewpoint, adipose tissue plays a critical role as an endocrine gland, secreting numerous factors with potent effects on the metabolism of distant tissues. These two new paradigms provide a framework to advance our understanding of the pathophysiol. of the insulin-resistance syndrome.
RE.CNT 118 THERE ARE 118 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L12 ANSWER 25 OF 60 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2002:907355 CAPLUS <<LOGINID::20070127>>
DN 138:300852
TI An adipocentric view of signaling and intracellular trafficking
AU Mora, Silvia; Pessin, Jeffrey E.
CS Department of Physiology and Biophysics, The University of Iowa, Iowa, IA, USA
SO Diabetes/Metabolism Research and Reviews (2002), 18(5), 345-356
CODEN: DMRRFM; ISSN: 1520-7552
PB John Wiley & Sons Ltd.
DT Journal; General Review
LA English
AB A review. Adipocytes have traditionally been considered to be the primary site for whole body energy storage mainly in the form of triglycerides and fatty acids. This occurs through the ability of insulin to markedly stimulate both glucose uptake and lipogenesis. Conventional wisdom held that defects in fuel partitioning into adipocytes either because of increased adipose tissue mass and/or increased lipolysis and circulating free fatty acids resulted in dyslipidemia, obesity, insulin resistance and perhaps diabetes. However, it has become increasingly apparent that loss of adipose tissue (lipodystrophies) in both animal models and humans also leads to metabolic disorders that result in severe states of insulin resistance and potential diabetes. These apparently opposite functions can be resolved by the establishment of adipocytes not only as a fuel storage depot but also as a critical endocrine organ that secretes a variety of signaling mols. into the circulation. Although the mol. function of these adipocyte-derived signals are poorly understood, they play a central role in the maintenance of energy homeostasis by regulating insulin secretion, insulin action, glucose and lipid metabolism, energy balance, host defense and reproduction The diversity of these secretory factors include enzymes (lipoprotein lipase (LPL) and adipsin), growth factors [vascular endothelial growth factor (VEGF)], cytokines (tumor necrosis factor-a, interleukin 6) and several other hormones involved in fatty acid and glucose metabolism (leptin, Acrp30,

resistin and acylation stimulation protein). Despite the large number of mols. secreted by adipocytes, our understanding of the pathways and mechanisms controlling intracellular trafficking and exocytosis in adipocytes is poorly understood. In this article, we will review the current knowledge of the trafficking and secretion processes that take place in adipocytes, focusing our attention on two of the best characterized adipokine mols. (leptin and adiponectin) and on one of the most intensively studied regulated membrane proteins, the GLUT4 glucose transporter.
RE.CNT 174 THERE ARE 174 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> D bib ABS l12 33-35, 41, 45, 49, 50-53, 55-60

L12 ANSWER 33 OF 60 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2002:786585 CAPLUS <<LOGINID::20070127>>
DN 138:83795
TI Regulation of adiponectin and leptin gene expression in white and brown adipose tissues: influence of b3-adrenergic agonists, retinoic acid, leptin and fasting
AU Zhang, Yi; Matheny, Michael; Zolotukhin, Sergei; Tumer, Nihal; Scarpace, Philip J.
CS Department of Veterans Affairs Medical Center, Geriatric Research, Education and Clinical Center, Gainesville, FL, 32608-1197, USA
SO Biochimica et Biophysica Acta, Molecular and Cell Biology of Lipids (2002), 1584(2-3), 115-122
CODEN: BBMLFG; ISSN: 1388-1981
PB Elsevier B.V.
DT Journal
LA English
AB Circulating adiponectin levels fall, whereas leptin levels rise with obesity, suggesting that regulation of these two adipocyte-derived hormones may be simultaneously influenced by common obesity-related factors. The authors examined adiponectin mRNA levels in WAT and in some instances, brown adipose tissue (BAT) following fasting and refeeding, acute and chronic administration of a b3-adrenergic agonist, acute treatment with retinoic acid (RA) and a glucocorticoid, and following chronic infusion of leptin and compared the expression of adiponectin to that of leptin in each circumstance. Serum concns. of adiponectin were also reported for most of the treatments. Fasting diminished and refeeding reversed both adiponectin and leptin gene expression. Peripheral injection of the b3-adrenergic agonist, CL316,243, suppressed both leptin and adiponectin expression in WAT. A small but significant reduction in adiponectin expression in BAT was also observed following this treatment. Although CL316,23 lowered serum leptin levels markedly, it did not affect serum adiponectin levels. A chronic 7-day infusion of CL316,243 resulted in an elevation of adiponectin expression in WAT and serum concns. in contrast to suppressions in both mRNA and serum levels of leptin by a similar treatment as previously reported. Chronic administration of leptin did not alter adiponectin synthesis in WAT compared to controls, but prevented the reduction in adiponectin synthesis associated with pair feeding. Food restriction through pair feeding also diminished adiponectin expression in BAT. Collectively, although leptin and adiponectin are inversely correlated with obesity, leptin does not appear to participate directly in adiponectin synthesis. The short-term regulation of the two adipokine expression in WAT is somewhat similar, perhaps subjective to common control of energy balance. The long-term regulation of adiponectin expression in WAT appears to be the opposite of that of leptin and may be more sensitive to changes in adiposity or insulin sensitivity.
RE.CNT 35 THERE ARE 35 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L12 ANSWER 34 OF 60 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2003:785040 CAPLUS <<LOGINID::20070127>>
DN 140:233198
TI Adiponectin and resistin
AU Ohuchi, Noriari; Funabashi, Toru; Matsuzawa, Yuji
CS Graduate School of Medicine, Osaka University, Japan
SO Bunshi Tonyobyogaku no Shinpo (2002) 53-59
CODEN: BTSHFO
PB Kanehara Shuppan
DT Journal; General Review
LA Japanese
AB A review. The topics discussed are (1) adipocytokines; (2) adipocyte-derived plasma protein adiponectin and its effects on atherogenesis suppression and improved insulin sensitivity; (3) resistin expression in relation to obesity and insulin resistance; and (4) other adipocytokines tumor necrosis factor-a (TNF-a) and leptin.

L12 ANSWER 35 OF 60 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
AN 2002:374089 BIOSIS <<LOGINID::20070127>>
DN PREV200200374089

TI Control of energy homeostasis and insulin action by adipocyte hormones: Leptin, acylation stimulating protein, and adiponectin.
AU Havel, Peter J. [Reprint author]
CS Department of Nutrition, University of California, Davis, One Shields Avenue, Davis, CA, 95616, USA
pjhavel@ucdavis.edu
SO Current Opinion in Lipidology, (February, 2002) Vol. 13, No. 1, pp. 51-59. print.
ISSN: 0957-9672.
DT Article
General Review; (Literature Review)
LA English
ED Entered STN: 3 Jul 2002
Last Updated on STN: 3 Jul 2002

L12 ANSWER 41 OF 60 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN
AN 2002317642 EMBASE <<LOGINID::20070127>>
TI Adiponectin enhances insulin action by decreasing ectopic fat deposition.
AU Ravussin E.
CS E. Ravussin, Pennington Biomedical Research Ctr., Health/Performance Enhancement Ctr., 6400 Perkins Rd., Baton Rouge, LA 70808 4124, United States. ravusse@pbrc.edu
SO Pharmacogenomics Journal, (2002) Vol. 2, No. 1, pp. 4-7. .
Refs: 16
ISSN: 1470-269X CODEN: PJHOAZ
CY United Kingdom
DT Journal; Article
FS 003 Endocrinology
022 Human Genetics
030 Pharmacology
037 Drug Literature Index
LA English
ED Entered STN: 19 Sep 2002
Last Updated on STN: 19 Sep 2002
DATA NOT AVAILABLE FOR THIS ACCESSION NUMBER

L12 ANSWER 45 OF 60 MEDLINE on STN
AN 2001666524 MEDLINE <<LOGINID::20070127>>
DN PubMed ID: 11712415
TI The molecular mechanisms by which PPAR gamma/RXR inhibitors improve insulin resistance.
AU Yamauchi T; Kadowaki T
CS Department of Metabolic Diseases, Graduate School of Medicine, University of Tokyo.
SO Nippon rinsho. Japanese journal of clinical medicine, (2001 Nov) Vol. 59, No. 11, pp. 2245-54. Ref: 22
Journal code: 0420546. ISSN: 0047-1852.
CY Japan
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
LA Japanese
FS Priority Journals
EM 200201
ED Entered STN: 20 Nov 2001
Last Updated on STN: 28 Jan 2002
Entered Medline: 25 Jan 2002
AB Potent activation of PPAR gamma by thiazolidinediones(TZD) increases TG content of WAT, thereby decreasing TG content of liver/muscle, leading to amelioration of insulin resistance at the expense of obesity.
Moderate reduction of PPAR gamma activity by PPAR gamma/RXR inhibitors decreases TG content of WAT/muscle/liver due to increased leptin and increase in fatty-acid combustion and decrease in lipogenesis, thereby ameliorating HF diet-induced obesity and insulin resistance.
Moreover, PPAR gamma/RXR inhibitors decrease lipogenesis in WAT, while TZD stimulate adipocyte differentiation and apoptosis, thereby both preventing adipocyte hypertrophy, which is associated with alleviation of insulin resistance presumably due to decreases in FFA, and TNF alpha, and upregulation of adiponectin. We conclude that although by different mechanisms, both PPAR gamma/RXR inhibitors and PPAR gamma agonist improve insulin resistance, which is associated with decreased TG content of muscle/liver and prevention of adipocyte hypertrophy.

L12 ANSWER 49 OF 60 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on
STN
AN 2001:397010 BIOSIS <<LOGINID::20070127>>
DN PREV200100397010
TI The fat-derived hormone adiponectin reverses insulin resistance associated with both lipoatrophy and obesity.
AU Yamauchi, T.; Kamon, J.; Waki, H.; Terauchi, Y.; Kubota, N.; Hara, K.; Mori, Y.; Ide, T.; Murakami, K.; Tsuboyama-Kasaoka, N.; Ezaki, O.; Akanuma, Y.; Gavrilova, O.; Vinson, C.; Reitman, M. L.; Kagechika, H.; Shudo, K.; Yoda, M.; Nakano, Y.; Tobe, K.; Nagai, R.; Kimura, S.; Tomita, M.; Froguel, P.; Kadowaki, T. [Reprint author]
CS Department of Internal Medicine, Graduate School of Medicine, University of Tokyo, Tokyo, Japan
kadowaki-3im@h.u-tokyo.ac.jp
SO Nature Medicine, (August, 2001) Vol. 7, No. 8, pp. 941-946.
print.
ISSN: 1078-8956.

DT Article
LA English
ED Entered STN: 22 Aug 2001
Last Updated on STN: 22 Feb 2002
AB Adiponectin is an adipocyte-derived hormone. Recent genome-wide scans have mapped a susceptibility locus for type 2 diabetes and metabolic syndrome to chromosome 3q27, where the gene encoding adiponectin is located. Here we show that decreased expression of adiponectin correlates with insulin resistance in mouse models of altered insulin sensitivity. Adiponectin decreases insulin resistance by decreasing triglyceride content in muscle and liver in obese mice. This effect results from increased expression of molecules involved in both fatty-acid combustion and energy dissipation in muscle. Moreover, insulin resistance in lipoatrophic mice was completely reversed by the combination of physiological doses of adiponectin and leptin, but only partially by either adiponectin or leptin alone. We conclude that decreased adiponectin is implicated in the development of insulin resistance in mouse models of both obesity and lipoatrophy. These data also indicate that the replenishment of adiponectin might provide a novel treatment modality for insulin resistance and type 2 diabetes.

L12 ANSWER 50 OF 60 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2002:34308 CAPLUS <<LOGINID::20070127>>
DN 137:150289
TI Insulin resistance and cytokines
AU Hirose, Hiroshi; Yajima, Ken; Yamamoto, Hiroyuki; Kawai, Toshihide; Ishii, Tatsuya; Fujita, Haruhisa; Seto, Yoshiko; Miyashita, Kiichi; Nishikai, Kanako; Hayashi, Keisuke; Kawabe, Hiroshi; Saito, Ikuo; Saruta, Takao
CS School of Medicine, Department of Internal Medicine, Keio University, Japan
SO Diabetes Frontier (2001), 12(5), 590-596
CODEN: DIFREZ; ISSN: 0915-6593
PB Medikaru Rebyusha
DT Journal; General Review
LA Japanese
AB A review on contributions of adipocytokines adipose-derived bioactive substances in the development of insulin resistance in obesity, diabetes, hypertension, and hyperlipidemia. Adipocytokines discussed are free fatty acids, tumor necrosis factor-a, leptin, adiponectin, and resistin.

L12 ANSWER 51 OF 60 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on
STN
AN 2001:540867 BIOSIS <<LOGINID::20070127>>
DN PREV200100540867
TI Physiological role of adipose tissue: White adipose tissue as an endocrine and secretory organ.
AU Trayhurn, Paul [Reprint author]; Beattie, John H.
CS Department of Medicine, University Clinical Departments, University of Liverpool, Liverpool, L69 3GA, UK
p_trayhurn@altavista.com
SO Proceedings of the Nutrition Society, (August, 2001) Vol. 60, No. 3, pp. 329-339. print.
CODEN: PNUSA4. ISSN: 0029-6651.
DT Article
General Review; (Literature Review)
LA English
ED Entered STN: 21 Nov 2001
Last Updated on STN: 25 Feb 2002
AB The traditional role attributed to white adipose tissue is energy storage, fatty acids being released when fuel is required. The metabolic role of white fat is, however, complex. For example, the tissue is needed for normal glucose homeostasis and a role in inflammatory processes has been proposed. A radical change in perspective followed the discovery of leptin; this critical hormone in energy balance is produced principally by white fat, giving the tissue an endocrine function. Leptin is one of a number of proteins secreted from white adipocytes, which include angiotensinogen, adipsin, acylation-stimulating protein, adiponectin, retinol-binding protein, tumour neorosis factor alpha, interleukin 6, plasminogen activator inhibitor-1 and tissue factor. Some of these proteins are inflammatory cytokines, some play a role in lipid metabolism, while others are involved in vascular haemostasis or the complement system. The effects of specific proteins may be autocrine or paracrine, or the site of action may be distant from adipose tissue. The most recently described adipocyte secretory proteins are fasting-induced adipose factor, a fibrinogen-angiopoietin-related protein, metallothionein and resistin. Resistin is an adipose tissue-specific factor which is reported to induce insulin resistance, linking diabetes to obesity. Metallothionein is a metal-binding and stress-response protein which may have an antioxidant role. The key challenges in establishing the secretory functions of white fat are to identify the complement of secreted proteins, to establish the role of each secreted protein, and to assess the pathophysiological consequences of changes in adipocyte protein production with alterations in adiposity (obesity, fasting, cachexia). There is already considerable evidence of links between increased production of some adipocyte factors and the metabolic and cardiovascular complications of obesity. In essence, white adipose tissue is a major secretory and endocrine organ involved in a range of functions beyond simple fat storage.

L12 ANSWER 56 OF 60 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on
STN
AN 2000:304930 BIOSIS <<LOGINID::20070127>>
DN PREV200000304930
TI Plasma concentrations of a novel, adipose-specific protein, adiponectin, in type 2 diabetic patients.
AU Hotta, Kikuko [Reprint author]; Funahashi, Tohru; Arita, Yukio; Takahashi, Masahiko; Matsuda, Morihiro; Okamoto, Yoshihisa; Iwahashi, Hiromi; Kuriyama, Hiroshi; Ouchi, Noriyuki; Maeda, Kazuhisa; Nishida, Makoto; Kihara, Shinji; Sakai, Naohiko; Nakajima, Tadahisa; Hasegawa, Kyoichi; Muraguchi, Masahiro; Ohmoto, Yasukazu; Nakamura, Tadashi; Yamashita, Shizuya; Hanafusa, Toshiaki; Matsuzawa, Yuji
CS Department of Internal Medicine and Molecular Science, Graduate School of Medicine, Osaka University, 2-2 Yamadaoka, Suita, Osaka, 565-0871, Japan
SO Arteriosclerosis Thrombosis and Vascular Biology, (June, 2000) Vol. 20, No. 6, pp. 1595-1599. print.
ISSN: 1079-5642.
DT Article
LA English
ED Entered STN: 19 Jul 2000
Last Updated on STN: 7 Jan 2002
AB Adiponectin is a novel, adipose-specific protein abundantly present in the circulation, and it has antiatherogenic properties. We analyzed the plasma adiponectin concentrations in age- and body mass index (BMI)-matched nondiabetic and type 2 diabetic subjects with and without coronary artery disease (CAD). Plasma levels of adiponectin in the diabetic subjects without CAD were lower than those in nondiabetic subjects (6.6+-0.4 versus 7.9+-0.5 mug/mL in men, 7.6+-0.7 versus 11.7+-1.0 mug/mL in women; P<0.001). The plasma adiponectin concentrations of diabetic patients with CAD were lower than those of diabetic patients without CAD (4.0+-0.4 versus 6.6+-0.4 mug/mL, P<0.001 in men; 6.3+-0.8 versus 7.6+-0.7 mug/mL in women). In contrast, plasma levels of leptin did not differ between diabetic patients with and without CAD. The presence of microangiopathy did not affect the plasma adiponectin levels in diabetic patients. Significant, univariate, inverse correlations were observed between adiponectin levels and fasting plasma insulin (r=-0.18, P<0.01) and glucose (r=-0.26, P<0.001) levels. In multivariate analysis, plasma insulin did not independently affect the plasma adiponectin levels. BMI, serum triglyceride concentration, and the presence of diabetes or CAD remained significantly related to plasma adiponectin concentrations. Weight reduction significantly elevated plasma adiponectin levels in the diabetic subjects as well as the nondiabetic subjects. These results suggest that the decreased plasma adiponectin concentrations in diabetes may be an indicator of macroangiopathy.

L12 ANSWER 57 OF 60 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2001:42054 CAPLUS <<LOGINID::20070127>>
DN 134:54607
TI The influence of the genes expressed in adipose tissue on diseases
AU Takahashi, Masahiko; Funahashi, Tohru
CS Dep. Intern. Med. Mol. Sci., Grad. Sch. Med., Osaka Univ., Japan
SO Horumon to Rinsho (2000), 48(12), 1055-1062
CODEN: HORIAE; ISSN: 0045-7167
PB Igaku no Sekaisha
DT Journal; General Review
LA Japanese
AB A review with 26 refs., on the pathol. of visceral fat syndrome, genes expressed in adipose tissues, and involvement of adipocytokines in the pathogenesis of coronary artery diseases, diabetes mellitus, hypertension, and other common diseases. Structure, distribution, and pathophysiol. functions of adiponectin/apM 1 (adipose most abundant gene transcript 1), plasminogen activator inhibitor 1, TNFa, leptin, PPARg, SREBP, and aquaporin adipose are discussed.

L12 ANSWER 58 OF 60 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on
STN
AN 2000:490437 BIOSIS <<LOGINID::20070127>>
DN PREV200000490558
TI Molecular mechanism of obesity-related diseases: Importance of adipocytokines.
AU Matsuzawa, Yuji [Reprint author]; Funahashi, Tohru; Kuriyama, Hiroshi; Kihara, Shinji
CS Department of Internal Medicine and Molecular Science, Graduate School of Medicine, Osaka University 2-2 B-5, Yamadaoka, Suita, Osaka, 565-0871, Japan
SO Imura, Hiroo; Kasuga, Masato; Nakao, Kazuwa. Int. Congr. Ser. - Excerpta Med., (1999) pp. 37-43. International Congress Series; Common disease: Genetic and pathogenetic aspects of multifactorial diseases. print.
Publisher: Elsevier Science B.V., Sara Burgerhartstraat 25, 1000 AE, Amsterdam, Netherlands. Series: International Congress Series.
Meeting Info.: Proceedings of the Uehara Memorial Foundation Symposium on Common Disease. Tokyo, Japan. June 30-July 02, 1999.
CODEN: EXMDA4. ISSN: 0531-5131. ISBN: 0-444-50200-9 (cloth).
DT Book
Conference; (Meeting)
Book; (Book Chapter)
Conference; (Meeting Paper)

L12 ANSWER 52 OF 60 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on
STN
AN 2001:564208 BIOSIS <<LOGINID::20070127>>
DN PREV200100564208
TI PPARgamma agonist and antagonist.
AU Kadowaki, Takashi [Reprint author]
CS Department of Metabolic Diseases, Graduate School of Medicine, University of Tokyo, Tokyo, 113-8655, Japan
kadowaki-3im@h.u-tokyo.ac.jp
SO Folia Pharmacologica Japonica, (November, 2001) Vol. 118, No. 5, pp. 321-326. print.
CODEN: NYKZAU. ISSN: 0015-5691.
DT Article
LA Japanese
ED Entered STN: 5 Dec 2001
Last Updated on STN: 25 Feb 2002
AB Peroxisome proliferator-activated receptor gamma (PPARgamma) is a ligand-activated transcription factor and functions as a heterodimer with a retinoid X receptor (RXR). Supraphysiological activation of PPARgamma by thiazolidinediones can reduce insulin resistance and hyperglycemia in type 2 diabetes, but these drugs can also cause weight gain. Quite unexpectedly, a moderate reduction of PPARgamma activity observed in heterozygous PPARgamma-deficient mice or the Pro 12 Ala polymorphism in human PPARgamma has been shown to prevent insulin resistance and obesity induced by a high-fat (HF) diet. We investigated whether functional antagonism toward PPARgamma/RXR could be used to treat obesity and type 2 diabetes. We show herein that moderate reduction of PPARgamma with an RXR antagonist or a PPARgamma antagonist decreases triglyceride (TG) content in white adipose tissue, skeletal muscle and liver. These inhibitors potentiate leptin's effects and stimulated adiponectin levels, which increases fatty acid combustion and energy dissipation, thereby ameliorating HF diet-induced obesity and insulin resistance. Paradoxically, severe reduction of PPARgamma by treatment of heterozygous PPARgamma-deficient mice with an RXR antagonist or a PPARgamma antagonist depletes white adipose tissue and markedly decreases leptin and adiponectin levels and energy dissipation, which increases TG content in skeletal muscle and the liver, thereby leading to the re-emergence of insulin resistance. Our data suggest that appropriate functional antagonism of PPARgamma/RXR may be a logical approach to protection against obesity and related diseases such as type 2 diabetes.

L12 ANSWER 53 OF 60 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2001:279651 CAPLUS <<LOGINID::20070127>>
DN 134:250319
TI Insulin resistance and visceral obesity
AU Nishida, Makoto; Funahashi, Tohru
CS Dep. Intern. Med. Mol. Sci., Grad. Sch. Med., Osaka Univ., Japan
SO Horumon to Rinsho (2001), 49(3), 227-233
CODEN: HORIAE; ISSN: 0045-7167
PB Igaku no Sekaisha
DT Journal; General Review
LA Japanese
AB A review with 37 refs., on the clin. importance of visceral fat syndrome, pathophysiol. functions of adipocytokines, structure and functions of adiponectin, mechanism of the induction of insulin resistance by visceral fat accumulation, action mechanisms of thiazolidine derivs. (PPARg agonists), roles of free fatty acids in insulin resistance, and involvement of adipocytokines (TNFa, leptin, and adiponectin) in insulin resistance.

L12 ANSWER 55 OF 60 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on
STN
AN 2001:441526 BIOSIS <<LOGINID::20070127>>
DN PREV200100441526
TI Replenishment of fat-derived hormone adiponectin reverses insulin resistance in lipoatrophic diabetes and type 2 diabetes.
AU Yamauchi, Toshimasa [Reprint author]; Kamon, Junji [Reprint author]; Terauchi, Yasuo [Reprint author]; Kubota, Naoto [Reprint author]; Waki, Hironori [Reprint author]; Mori, Yasumichi [Reprint author]; Hara, Kazuo [Reprint author]; Akanuma, Yasuo [Reprint author]; Kimura, Satoshi [Reprint author]; Tobe, Kazuyuki [Reprint author]; Yoda, Madoka [Reprint author]; Tomita, Motowo [Reprint author]; Froguel, Philippe [Reprint author]; Kadowaki, Takashi [Reprint author]
CS Tokyo, Japan
SO Diabetes, (June, 2001) Vol. 50, No. Supplement 2, pp. A70. print.
Meeting Info.: 61st Scientific Sessions of the American Diabetes Association. Philadelphia, Pennsylvania, USA. June 22-26, 2001. American Diabetes Association.
CODEN: DIAEAZ. ISSN: 0012-1797.
DT Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LA English
ED Entered STN: 19 Sep 2001
Last Updated on STN: 22 Feb 2002

LA English
ED Entered STN: 15 Nov 2000
Last Updated on STN: 10 Jan 2002

L12 ANSWER 59 OF 60 CAPLUS COPYRIGHT 2007 ACS on STN
AN 1999:687090 CAPLUS <<LOGINID::20070127>>
DN 132:32236
TI Cell biology of visceral fat
AU Hotta, Kikuko; Matsuzawa, Yuji
CS Department of Internal Medicine and Molecular Science, Graduate School of Medicine, Osaka University, Yamadaoka, Suita-shi, Osaka, 565-0871, Japan
SO Nihon Yukagakkaishi (1999), 48(10), 963-970
CODEN: NIYUFC; ISSN: 1341-8327
PB Nihon Yukagaku Gakkai
DT Journal; General Review
LA Japanese
AB A review with 49 refs. Adipose tissue is a source of passively stored excess energy. Adipose tissue has been found to secrete various biol. active adipocytokines such as leptin, plasminogen activator inhibitor (PAI)-1 and tumor necrosis factor (TNF) a, which affect homeostasis throughout the body. Plasma adipocytokines increase in obesity and the accumulation of fat, especially visceral fat, may serve to create greater insulin resistance or thrombotic tendency in obesity, through enhanced secretion of the above compds. In search for genes expressed in adipose tissue, novel adipose-specific genes, adiponectin and aquaporin adipose were isolated in the present study. Adiponectin had a collagen-like sequence and was secreted into blood. Plasma adiponectin paradoxically decreased in obesity, but was expressed exclusively in adipose tissue. Aquaporin adipose is also highly expressed in adipose tissue. This new water channel transports glycerol as well as water, suggesting aquaporin adipose to possibly be essential to glycerol metabolism in adipocytes. The finding of genes specifically expressed in visceral fat and new adipocytokines should facilitate clarification of the mechanism for the development and complications of visceral fat accumulation.

L12 ANSWER 60 OF 60 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2000:101167 CAPLUS <<LOGINID::20070127>>
DN 133:28940
TI Molecular mechanism of obesity-related diseases: importance of adipocytokines
AU Matsuzawa, Yuji; Funahashi, Tohru; Kuriyama, Hiroshi; Kihara, Shinji
CS Department of Internal Medicine and Molecular Science, Graduate School of Medicine, Osaka University, Suita, 565-0871, Japan
SO International Congress Series (1999), 1181(Common Disease: Genetic and Pathogenetic Aspects of Multifactorial Diseases), 37-43
CODEN: EXMDA4; ISSN: 0531-5131
PB Elsevier Science B.V.
DT Journal; General Review
LA English
AB A review, with 12 refs. Obesity is a major cause of common human disorders including diabetes mellitus, hyperlipidemia, hypertension, and atherosclerotic vascular disease. Recent studies on adipocyte biol. have revealed that adipose tissue is not simply an energy storage organ but also an endocrine organ secreting a variety of bioactive substances called "adipocytokines" which affect biol. function of each target organ. For example, TNF-a from adipose tissues is one of the key factors for the development of insulin resistance. Leptin is another famous adipocytokines, which have an important role in controlling appetite and energy expenditure. To clarify the mol. characteristics of adipose tissue, a systemic anal. of expressed genes was performed using large-scale-random sequencing and revealed that adipose tissue, especially visceral adipose tissue expressed numerous genes for secretory proteins (.apprx.30% and .apprx.20% of the total genes in visceral and s.c. adipose tissue, resp.). Among these secretory proteins, active genes reputedly related to atherogenesis such as plasminogen activator inhibitor-1 (PAI-1) and heparin-binding EGF-like growth factor (HB-EGF) were found in the library. PAI-1, a regulator of fibrinolytic system, was overexpressed in the visceral adipose tissue in an animal model of obesity. Plasma levels of PAI-1 were closely correlated with visceral adiposity in human subjects. A novel adipose-specific collagen-like mol. named adiponectin was found. This novel mol. is suggested to have an anti-atherogenic property such as inhibition of smooth muscle cell proliferation and inhibition of adhesion mol. expression in endothelial cells, etc. The plasma levels of adiponectin unlike leptin were neg. correlated with body mass indexes. Thus, adipose tissue acts as an endocrine organ secreting a variety of bioactive substances, adipocytokines. Hypersecretion of adipocytokines such as PAI-1 or TNF-a and hyposecretion of those such as adiponectin in obese state may relate to the pathogenesis of obesity-related diseases including diabetes mellitus and vascular disease.
RE.CNT 12 THERE ARE 12 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> D bib ABS L14 9, 14

L14 ANSWER 9 OF 15 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
AN 2002:266783 BIOSIS <<LOGINID::20070127>>

DN PREV200200266783
TI The mechanisms by which PPARgamma regulates insulin sensitivity.
AU Yamauchi, Toshimasa [Reprint author]; Kadowaki, Takashi [Reprint author]
CS Dept. of Metabolic Disease, Graduate Sch. of Med., Univ. of Tokyo, Tokyo, Japan
SO Japanese Journal of Pharmacology, (2002) Vol. 88, No. Supplement 1, pp. 24P. print.
Meeting Info.: 75th Annual Meeting of the Japanese Pharmacological Society. Kumamoto, Japan. March 13-15, 2002. Japanese Pharmacological Society.
CODEN: JJPAAZ. ISSN: 0021-5198.
DT Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LA English
ED Entered STN: 1 May 2002
Last Updated on STN: 1 May 2002

L14 ANSWER 14 OF 15 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
AN 2001:187493 BIOSIS <<LOGINID::20070127>>
DN PREV200100187493
TI Leptin - signals and secretions from white adipose tissue.
AU Trayhurn, Paul [Reprint author]; Beattie, John H. [Reprint author]; Rayner, D. Vernon [Reprint author]
CS Rowett Research Institute, Bucksburn, Aberdeen, AB21 9SB, UK
SO Heldmaier, Gerhard; Klingenspor, Martin. (2000) pp. 459-469.
Life in the cold. print.
Publisher: Springer-Verlag GmbH and Co. KG, Heidelberger Platz 3, D-14197, Berlin, Germany; Springer-Verlag New York Inc., 175 Fifth Avenue, New York, NY, 10010-7858, USA.
Meeting Info.: Eleventh International Hibernation Symposium. Jungholz, Austria. August 13-18, 2000.
ISBN: 3-540-67410-1 (cloth).
DT Book
Conference; (Meeting)
Book; (Book Chapter)
Conference; (Meeting Paper)
LA English
ED Entered STN: 20 Apr 2001
Last Updated on STN: 18 Feb 2002

=> D bib Abs L16 1, 3, 10, 14, 30, 39, 42, 43, 50, 65, 73-75,91, 126, 157, 167, 178, 183

L16 ANSWER 1 OF 191 MEDLINE on STN
AN 2002669523 MEDLINE <<LOGINID::20070127>>
DN PubMed ID: 12429885
TI Resistin and adiponectin--of mice and men.
AU Stumvoll Michael; Haring Hans
SO Obesity research, (2002 Nov) Vol. 10, No. 11, pp. 1197-9. Ref: 26
Journal code: 9305691. ISSN: 1071-7323.
CY United States
DT Editorial
General Review; (REVIEW)
LA English
FS Priority Journals
EM 200305
ED Entered STN: 14 Nov 2002
Last Updated on STN: 14 May 2003
Entered Medline: 13 May 2003

L16 ANSWER 3 OF 191 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
AN 2003:32810 BIOSIS <<LOGINID::20070127>>
DN PREV200300032810
TI Increased fat intake, impaired fat oxidation, and failure of fat cell proliferation result in ectopic fat storage, insulin resistance, and type 2 diabetes mellitus.
AU Ravussin, Eric [Reprint Author]; Smith, Steven R.
CS Pennington Biomedical Research Center, 6400 Perkins Road, Baton Rouge, LA, 70808-4124, USA
ravusse@pbrc.edu
SO Klimes, Iwar [Editor, Reprint Author]; Sebokova, Elena [Editor]; Howard, Barbara V. [Editor]; Ravussin, Eric [Editor]. (2002) pp. 363-378. Lipids and insulin resistance: The role of fatty acid metabolism and fuel partitioning. print.
Publisher: New York Academy of Sciences, 2 East 63rd Street, New York, NY, 10021, USA. Series: Annals of the New York Academy of Sciences.
Meeting Info.: Fourth International Smolenice Insulin Symposium on Lipids and Insulin Resistance: The Role of Fatty Acid Metabolism and Fuel Partitioning. Smolenice, Slovakia. August 29-September 02, 2001.
ISSN: 0077-8923 (ISSN print). ISBN: 1-57331-368-8 (cloth), 1-57331-369-6 (paper).
DT Book; (Book Chapter)
Conference; (Meeting)
Conference; (Meeting Paper)
LA English
ED Entered STN: 8 Jan 2003

Last Updated on STN: 8 Jan 2003

L16 ANSWER 10 OF 191 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2002:775209 CAPLUS <<LOGINID::20070127>>
DN 138:37209
TI Removal of visceral fat prevents insulin resistance and glucose intolerance of aging: an adipokine-mediated process?
AU Gabriely, Ilan; Ma, Xiao Hui; Yang, Xiao Man; Atzmon, Gil; Rajala, Michael W.; Berg, Anders H.; Scherer, Phillip; Rossetti, Luciano; Barzilai, Nir
CS Diabetes Research and Training Center and Division of Endocrinology, Department of Medicine, Institute for Aging Research, Albert Einstein College of Medicine, Bronx, NY, 10461, USA
SO Diabetes (2002), 51(10), 2951-2958
CODEN: DIAEAZ; ISSN: 0012-1797
PB American Diabetes Association
DT Journal
LA English
AB Age-dependent changes in insulin action and body fat distribution are risk factors for the development of type 2 diabetes
. To examine whether the accumulation of visceral fat (VF) could play a direct role in the pathophysiol. of insulin resistance and type 2 diabetes, we monitored insulin action, glucose tolerance, and the expression of adipo-derived peptides after surgical removal of VF in aging (20-mo-old) F344/Brown Norway (FBN) and in Zucker Diabetic Fatty (ZDF) rats. As expected, peripheral and hepatic insulin action were markedly impaired in aging FBN rats, and extraction of VF (accounting for .apprx.18% of their total body fat) was sufficient to restore peripheral and hepatic insulin action to the levels of young rats. When examined at the mechanistic level, removal of VF in ZDF rats prevented the progressive decrease in insulin action and delayed the onset of diabetes, but VF extraction did not alter plasma free fatty acid levels. However, the expression of tumor necrosis factor-a and leptin in s.c. (SC) adipose tissue were markedly decreased after VF removal (by approx. three- and twofold, resp.). Finally, extracted VF retained .apprx.15-fold higher resistin mRNA compared with SC fat. Our data suggest that insulin resistance and the development of diabetes can be significantly reduced in aging rats by preventing the age-dependent accumulation of VF. This study documents a cause-and-effect relationship between VF and major components of the metabolic syndrome.
RE.CNT 39 THERE ARE 39 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L16 ANSWER 14 OF 191 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on
STN DUPLICATE 8

AN 2002:416819 BIOSIS <<LOGINID::20070127>>
DN PREV200200416819
TI Association of adiponectin mutation with type 2 diabetes: A candidate gene for the insulin resistance syndrome.
AU Kondo, Hidehiko; Shimomura, Iichiro; Matsukawa, Yuko; Kumada, Masahiro; Takahashi, Masahiko; Matsuda, Morihiro; Ouchi, Noriyuki; Kihara, Shinji; Kawamoto, Toshiharu; Sumitsuji, Satoru; Funahashi, Tohru [Reprint author]; Matsuzawa, Yuji
CS Department of Internal Medicine and Molecular Science, Osaka University Graduate School of Medicine, B5 2-2, Yamadaoka, Suita, Osaka, 565-0871, Japan
tohru@imed2.med.osaka-u.ac.jp
SO Diabetes, (July, 2002) Vol. 51, No. 7, pp. 2325-2328. print.
CODEN: DIAEAZ. ISSN: 0012-1797.
DT Article
LA English
ED Entered STN: 31 Jul 2002
Last Updated on STN: 31 Jul 2002
AB Adiponectin, also referred to as AdipoQ or ACRP30, is a plasma protein produced and secreted exclusively from adipose tissue. The protein contains a collagen-like domain and a C1q-like globular domain. A protease-generated globular segment enhances fatty acid oxidation in muscles, thereby modulating lipid and glucose metabolism. Plasma adiponectin levels are inversely correlated with the severity of insulin resistance. A recent genome-wide scan study mapped a susceptibility locus for type 2 diabetes and the metabolic syndrome to chromosome 3q27, where the adiponectin gene is located. Here, we screened Japanese patients with type 2 diabetes and age- and BMI-matched nondiabetic control subjects for mutations in adiponectin gene. We identified four missense mutations (R112C, I164T, R221S, and H241P) in the globular domain. Among these mutations, the frequency of I164T mutation was significantly higher in type 2 diabetic patients than in age- and BMI-matched control subjects ($P < 0.01$). Furthermore, plasma adiponectin concentrations of subjects carrying I164T mutation were lower than those of subjects without the mutation. All the subjects carrying I164T mutation showed some feature of metabolic syndrome, including hypertension, hyperlipidemia, diabetes, and atherosclerosis. Our findings suggest that I164T mutation is associated with low plasma adiponectin concentration and type 2 diabetes.

L16 ANSWER 30 OF 191 CAPLUS COPYRIGHT 2007 ACS on STN DUPLICATE 13
AN 2003:275394 CAPLUS <<LOGINID::20070127>>

DN 138:399418
TI Adiponectin - a link between obesity, atherosclerosis and diabetes
AU Sieminska, Lucyna; Marek, Bogdan; Kajdaniuk, Dariusz; Kos-Kudla, Beata; Czernecka, Dagmara
CS Zakl. Patofizjol., Katedra Patofizjol. i Endokrynol., Sl. Akad. Medyczna, Zabrze, Pol.
SO Polskie Archiwum Medycyny Wewnetrznej (2002), 108(6), 1245-1251
CODEN: PAMWAL; ISSN: 0032-3772
PB Wydawnictwo Medyczne Urban & Partner
DT Journal; General Review
LA Polish
AB A review. The topics include the biochem. of adiponectins, their cellular physiol., and possible roles in pathogenesis of obesity, atherosclerosis, and diabetes mellitus in humans.

L16 ANSWER 39 OF 191 CAPLUS COPYRIGHT 2007 ACS on STN DUPLICATE 19
AN 2003:65783 CAPLUS <<LOGINID::20070127>>
DN 138:265785
TI Adipose tissue hormones
AU Guerre-Millo, M.
CS Centre de Recherche des Cordeliers, Universite Pierre et Marie Curie, Paris, 75006, Fr.
SO Journal of Endocrinological Investigation (2002), 25(10), 855-861
CODEN: JEIND7; ISSN: 0391-4097
PB Editrice Kurtis s.r.l.
DT Journal; General Review
LA English
AB A review. It is now widely accepted that white adipose tissue (WAT) secretes a number of peptide hormones, including leptin, several cytokines, adipsin and acylation-stimulating protein (ASP), angiotensinogen, plasminogen activator inhibitor-1 (PAI-1), adiponectin, resistin etc., and also produces steroids hormones. This newly discovered secretory function has shifted the authors' view of WAT, which is no longer considered only an energy storage tissue but a major endocrine organ, at the heart of a complex network influencing energy homeostasis, glucose and lipid metabolism, vascular homeostasis, immune response and even reproduction Virtually all known adipose secreted proteins are dysregulated when the WAT mass is markedly altered, either increased in the obese state or decreased in lipoatrophy. This strongly implicates adiposesecreted products in the ethiopathol. and/or complications of both obesity and cachexia. This review discusses the physiol. relevance of adipose secretion by focusing on protein and steroid hormones. Regulation of WAT secretion by the major regulatory factors impinging on the adipocytes, i.e., insulin, glucocorticoids, catecholamines and thiazolidinediones (TZD) will be addressed. The rationale for therapeutic strategies aimed at compensating adverse effects resulting from overprodn. or lack of a specific adipose secretory product will be discussed.
RE.CNT 53 THERE ARE 53 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L16 ANSWER 42 OF 191 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2002:453341 CAPLUS <<LOGINID::20070127>>
DN 137:61015
TI Obesity and adipo-science
AU Yamauchi, Toshimasa; Kadowaki, Takashi
CS Grad. Sch. Med., The Univ. Tokyo, Japan
SO Rinsho Eiyo (2002), 100(6, Rinjizokango), 745-750
CODEN: RNEYAW; ISSN: 0485-1412
PB Ishiyaku Shuppan
DT Journal; General Review
LA Japanese
AB A review on improvement of insulin resistance by PPARg agonists, thiazolidine derivs., via acceleration of adipocyte differentiation and apoptosis, functions of PPARg as a thrift gene, PPARg2 gene polymorphism and type 2 diabetes mellitus, treatment of type 2 diabetes mellitus by PPARg inhibitors, regulation of insulin sensitivity by PPARg, and role of adiponectin in regulation of insulin sensitivity.

L16 ANSWER 43 OF 191 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2002:588796 CAPLUS <<LOGINID::20070127>>
DN 138:130470
TI Tailor-made medicine for obesity
AU Hotta, Kikuo
CS SNP Research Center, Institute of Physical and Chemical Research, Japan
SO Igaku no Ayumi (2002), 201(9), 725-728
CODEN: IGAYAY; ISSN: 0039-2359
PB Ishiyaku Shuppan
DT Journal; General Review
LA Japanese
AB A review, discussing the development of tailor-made medicine for obesity with regards to genomes and SNP related to adiponectins and adipocytokines.

L16 ANSWER 50 OF 191 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN DUPLICATE 23
AN 2002397931 EMBASE <<LOGINID::20070127>>
TI Diabetes, obesity, and Acrp30/adiponectin.

AU Hug C.; Lodish H.F.
CS Dr. H.F. Lodish, Whitehead Inst. for Biomed. Research, 9 Cambridge Center, Cambridge, MA 02142, United States. lodish@wi.mit.edu
SO BioTechniques, (2002) Vol. 33, No. 3, pp. 654-662. .
Refs: 33
ISSN: 0736-6205 CODEN: BTNQDO
CY United States
DT Journal; General Review
FS 003 Endocrinology
029 Clinical Biochemistry
LA English
ED Entered STN: 2 Dec 2002
Last Updated on STN: 2 Dec 2002
DATA NOT AVAILABLE FOR THIS ACCESSION NUMBER

L16 ANSWER 65 OF 191 CAPLUS COPYRIGHT 2007 ACS on STN DUPLICATE 33
AN 2002:832209 CAPLUS <<LOGINID::20070127>>
DN 138:117693
TI Adiponectin - its role in metabolism and beyond
AU Stefan, N.; Stumvoll, M.
CS Clinical Nutrition and Metabolism Section, NIDDK, NIH, Phoenix, AZ, 85016, USA
SO Hormone and Metabolic Research (2002), 34(9), 469-474
CODEN: HMMRA2; ISSN: 0018-5043
PB Georg Thieme Verlag
DT Journal; General Review
LA English
AB A review. Adiponectin is a recently identified adipose tissue-derived protein (adipocytokine) with important metabolic effects. It is exclusively expressed in adipose tissue and released into the circulation. Adiponectin expression and/or secretion is increased by insulin like growth factor-1 and ionomycin, and decreased by tumor necrosis factor-a, glucocorticoids, b-adrenergic agonists and cAMP. Data for insulin are somewhat inconclusive. Moreover, adiponectin expression and secretion are increased by activators of peroxisome proliferator-activated receptor (PPAR)-g. Besides inhibiting inflammatory pathways, recombinant adiponectin increases insulin sensitivity and improves glucose tolerance in various animal models. This insulin-sensitizing effect appears to be mostly attributable to enhanced suppression of glucose production, but beneficial effects on muscle cannot be excluded. In humans, plasma adiponectin concns. exceed those of any other hormone by a thousand times; they decrease with obesity and are pos. associated with whole-body insulin sensitivity. Therefore, low adiponectin may contribute to the decrease in whole-body insulin sensitivity that accompanies obesity. Furthermore, there is increasing evidence that genetic variants in the adiponectin gene itself and/or in genes encoding adiponectin-regulatory proteins - such as PPAR-g - may be associated with hypoadiponectinemia, insulin resistance and type 2 diabetes. This suggests that adiponectin may reflect PPAR-g activity in vivo. Finally, reversal or alleviation of hypoadiponectinemia may represent a target for development of drugs improving insulin sensitivity and glucose tolerance.
RE.CNT 58 THERE ARE 58 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L16 ANSWER 73 OF 191 CAPLUS COPYRIGHT 2007 ACS on STN DUPLICATE 35
AN 2002:587011 CAPLUS <<LOGINID::20070127>>
DN 137:382874
TI Increased fat intake, impaired fat oxidation, and failure of fat cell proliferation result in ectopic fat storage, insulin resistance, and type 2 diabetes mellitus
AU Ravussin, Eric; Smith, Steven R.
CS Pennington Biomedical Research Center, Baton Rouge, LA, 70808-4124, USA
SO Annals of the New York Academy of Sciences (2002), 967(Lipids and Insulin Resistance), 363-378
CODEN: ANYAA9; ISSN: 0077-8923
PB New York Academy of Sciences
DT Journal; General Review
LA English
AB A review. It is widely accepted that increasing adiposity is associated with insulin resistance and increased risk of type 2 diabetes. The predominant paradigm used to explain this link is the portal/visceral hypothesis. This hypothesis proposes that increased adiposity, particularly in the visceral depots, leads to increased free fatty acid flux and inhibition of insulin action via Randle's effect in insulin-sensitive tissues. Recent data do not entirely support this hypothesis. As such, two new paradigms have emerged that may explain the established links between adiposity and disease. Three lines of evidence support the ectopic fat storage syndrome. First, failure to develop adequate adipose tissue mass in either mice or humans, also known as lipodystrophy, results in severe insulin resistance and diabetes. This is thought to be the result of ectopic storage of lipid into liver, skeletal muscle, and the pancreatic insulin-secreting beta cell. Second, most obese patients also shunt lipid into the skeletal muscle, the liver, and probably the beta cell. The importance of this finding is exemplified by several studies demonstrating that the degree of lipid infiltration into skeletal muscle and liver correlates highly with insulin resistance. Third, increased fat cell size is highly associated with insulin resistance

and the development of diabetes. Increased fat cell size may represent the failure of the adipose tissue mass to expand and thus to accommodate an increased energy influx. Taken together, these three observations support the acquired lipodystrophy hypothesis as a link between adiposity and insulin resistance. The endocrine paradigm developed in parallel with the ectopic fat storage syndrome hypothesis. Adipose tissue secretes a variety of endocrine hormones, such as leptin, interleukin-6, angiotensin II, adiponectin (also called ACRP30 and adipoQ), and resistin. From this viewpoint, adipose tissue plays a critical role as an endocrine gland, secreting numerous factors with potent effects on the metabolism of distant tissues. These two new paradigms provide a framework to advance our understanding of the pathophysiol. of the insulin-resistance syndrome.

RE.CNT 118 THERE ARE 118 CITED REFERENCES AVAILABLE FOR THIS RECORD

ALL CITATIONS AVAILABLE IN THE RE FORMAT

L16 ANSWER 74 OF 191 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2002:578380 CAPLUS <<LOGINID::20070127>>
DN 138:87270
TI Adiponectin - antidiabetic and antiatherogenic adipocytokine
AU Shimomura, Iichiro; Hunahashi, Toru; Kihara, Shinji; Matsuzawa, Yuji
CS Dep. Internal Med. Molecular Sci., Grad. Sch. Med., Osaka Univ., Suita, 565-0871, Japan
SO Naibunpi, Tonyobyoka (2002), 14(4), 361-366
CODEN: NATOFF; ISSN: 1341-3724
PB Kagaku Hyoronsha
DT Journal; General Review
LA Japanese
AB A review, on the concept of adipocytokines, especially adiponectin, in lipodystrophy and obesity; adiponectin as adipocyte-specific hormones; and pathophysiol. roles of adiponectin in diabetes, atherosclerosis, and metabolic disorders.

L16 ANSWER 75 OF 191 CAPLUS COPYRIGHT 2007 ACS on STN DUPLICATE 36
AN 2002:907355 CAPLUS <<LOGINID::20070127>>
DN 138:300852
TI An adipocentric view of signaling and intracellular trafficking
AU Mora, Silvia; Pessin, Jeffrey E.
CS Department of Physiology and Biophysics, The University of Iowa, Iowa, IA, USA
SO Diabetes/Metabolism Research and Reviews (2002), 18(5), 345-356
CODEN: DMRRFM; ISSN: 1520-7552
PB John Wiley & Sons Ltd.
DT Journal; General Review
LA English
AB A review. Adipocytes have traditionally been considered to be the primary site for whole body energy storage mainly in the form of triglycerides and fatty acids. This occurs through the ability of insulin to markedly stimulate both glucose uptake and lipogenesis. Conventional wisdom held that defects in fuel partitioning into adipocytes either because of increased adipose tissue mass and/or increased lipolysis and circulating free fatty acids resulted in dyslipidemia, obesity, insulin resistance and perhaps diabetes. However, it has become increasingly apparent that loss of adipose tissue (lipodystrophies) in both animal models and humans also leads to metabolic disorders that result in severe states of insulin resistance and potential diabetes. These apparently opposite functions can be resolved by the establishment of adipocytes not only as a fuel storage depot but also as a critical endocrine organ that secretes a variety of signaling mols. into the circulation. Although the mol. function of these adipocyte-derived signals are poorly understood, they play a central role in the maintenance of energy homeostasis by regulating insulin secretion, insulin action, glucose and lipid metabolism, energy balance, host defense and reproduction The diversity of these secretory factors include enzymes (lipoprotein lipase (LPL) and adipsin), growth factors [vascular endothelial growth factor (VEGF)], cytokines (tumor necrosis factor-a, interleukin 6) and several other hormones involved in fatty acid and glucose metabolism (leptin, Acrp30, resistin and acylation stimulation protein). Despite the large number of mols. secreted by adipocytes, our understanding of the pathways and mechanisms controlling intracellular trafficking and exocytosis in adipocytes is poorly understood. In this article, we will review the current knowledge of the trafficking and secretion processes that take place in adipocytes, focusing our attention on two of the best characterized adipokine mols. (leptin and adiponectin) and on one of the most intensively studied regulated membrane proteins, the GLUT4 glucose transporter.

RE.CNT 174 THERE ARE 174 CITED REFERENCES AVAILABLE FOR THIS RECORD

ALL CITATIONS AVAILABLE IN THE RE FORMAT

L16 ANSWER 91 OF 191 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2002:378204 CAPLUS <<LOGINID::20070127>>
DN 137:308016
TI The role of adiponectin in obesity, insulin resistance, and type 2 diabetes. The fat-derived hormone adiponectin reverses insulin resistance associated with both lipoatrophy and obesity
AU Yamauchi, Toshimasa; Kadowaki, Takashi
CS Department of Metabolic Diseases, Graduate School of Medicine, University of Tokyo, Tokyo, 113-8655, Japan
SO Naibunpi, Tonyobyoka (2002), 14(2), 172-179
CODEN: NATOFF; ISSN: 1341-3724

PB Kagaku Hyoronsha
DT Journal; General Review
LA Japanese
AB A review, on roles of adiponectin in regulation of insulin sensitivity, discussing adiponectin expression and insulin sensitivity, adiponectin gene as the major disease-sensitive gene in Japanese population with type 2 diabetes; adiponectin as the major insulin sensitive hormone derived from white adipocytes; and adiponectin deficiency induction of obesity and type 2 diabetes and adiponectin supplement in improvement of insulin resistance.

L16 ANSWER 126 OF 191 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2002:16380 CAPLUS <<LOGINID::20070127>>
DN 137:138062
TI The role of adiponectin in obesity, insulin resistance, and type 2 diabetes: The fat-derived hormone adiponectin reverses insulin resistance associated with both lipoatrophy and obesity
AU Yamauchi, Toshimasa; Kadowaki, Takashi
CS Department of Metabolic Diseases, Graduate School of Medicine, University of Tokyo, Japan
SO Jikken Igaku (2001), 19(17), 2301-2305
CODEN: JIIGEF; ISSN: 0288-5514
PB Yodosha
DT Journal; General Review
LA Japanese
AB A review discussing increased adiponectin expression in heterozygous PPARg deficiency with improved insulin sensitivity; genetic variations in the adiponectin gene associated with increased risk of type 2 diabetes in Japanese population; fat-derived adiponectin as insulin sensitive hormone; and insulin resistance induced by adiponectin deficiency.

L16 ANSWER 157 OF 191 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
AN 2001:441526 BIOSIS <<LOGINID::20070127>>
DN PREV200100441526
TI Replenishment of fat-derived hormone adiponectin reverses insulin resistance in lipoatrophic diabetes and type 2 diabetes.
AU Yamauchi, Toshimasa [Reprint author]; Kamon, Junji [Reprint author]; Terauchi, Yasuo [Reprint author]; Kubota, Naoto [Reprint author]; Waki, Hironori [Reprint author]; Mori, Yasumichi [Reprint author]; Hara, Kazuo [Reprint author]; Akanuma, Yasuo [Reprint author]; Kimura, Satoshi [Reprint author]; Tobe, Kazuyuki [Reprint author]; Yoda, Madoka [Reprint author]; Tomita, Motowo [Reprint author]; Froguel, Philippe [Reprint author]; Kadowaki, Takashi [Reprint author]
CS Tokyo, Japan
SO Diabetes, (June, 2001) Vol. 50, No. Supplement 2, pp. A70.
print.
Meeting Info.: 61st Scientific Sessions of the American Diabetes Association. Philadelphia, Pennsylvania, USA. June 22-26, 2001. American Diabetes Association.
CODEN: DIAEAZ. ISSN: 0012-1797.
DT Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LA English
ED Entered STN: 19 Sep 2001
Last Updated on STN: 22 Feb 2002

L16 ANSWER 167 OF 191 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2000:777806 CAPLUS <<LOGINID::20070127>>
DN 133:308338
TI Adipocytokines
AU Yokota, Takafumi; Takahashi, Masahiko; Funahashi, Tohru
CS Grad. Sch. Med., Osaka Univ., Japan
SO Ensho to Men'eki (2000), 8(6), 624-629
CODEN: ENMEFA; ISSN: 0918-8371
PB Sentan Igakusha
DT Journal; General Review
LA Japanese
AB A review with 11 refs., on the expression of adipocytokines in visceral fat and their involvement in obesity complications, enhanced expression of PAI-1 in visceral fat, and structure and pathophysiol. functions of adiponectin. The decrease of adiponectin expression in humans with obesity and coronary artery diseases, and suppression of the proliferation and functions of macrophages by adiponectin are discussed.

L16 ANSWER 178 OF 191 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
AN 2000:490437 BIOSIS <<LOGINID::20070127>>
DN PREV200000490558
TI Molecular mechanism of obesity-related diseases: Importance of adipocytokines.
AU Matsuzawa, Yuji [Reprint author]; Funahashi, Tohru; Kuriyama, Hiroshi; Kihara, Shinji

CS Department of Internal Medicine and Molecular Science, Graduate School of Medicine, Osaka University 2-2 B-5, Yamadaoka, Suita, Osaka, 565-0871, Japan
SO Imura, Hiroo; Kasuga, Masato; Nakao, Kazuwa. Int. Congr. Ser. - Excerpta Med., (1999) pp. 37-43. International Congress Series; Common disease: Genetic and pathogenetic aspects of multifactorial diseases. print.
Publisher: Elsevier Science B.V., Sara Burgerhartstraat 25, 1000 AE, Amsterdam, Netherlands. Series: International Congress Series.
Meeting Info.: Proceedings of the Uehara Memorial Foundation Symposium on Common Disease. Tokyo, Japan. June 30-July 02, 1999.
CODEN: EXMDA4. ISSN: 0531-5131. ISBN: 0-444-50200-9 (cloth).
DT Book
Conference; (Meeting)
Book; (Book Chapter)
Conference; (Meeting Paper)
LA English
ED Entered STN: 15 Nov 2000
Last Updated on STN: 10 Jan 2002

L16 ANSWER 183 OF 191 MEDLINE on STN
AN 1999240218 MEDLINE <<LOGINID::20070127>>
DN PubMed ID: 10225688
TI Role of adipocytokines on the pathogenesis of atherosclerosis in visceral obesity.
AU Funahashi T; Nakamura T; Shimomura I; Maeda K; Kuriyama H; Takahashi M; Arita Y; Kihara S; Matsuzawa Y
CS The Second Department of Internal Medicine, Osaka University Medical School, Suita.
SO Internal medicine (Tokyo, Japan), (1999 Feb) Vol. 38, No. 2, pp. 202-6. Ref: 14
Journal code: 9204241. ISSN: 0918-2918.
CY Japan
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
LA English
FS Priority Journals
EM 199906
ED Entered STN: 12 Jul 1999
Last Updated on STN: 12 Jul 1999
Entered Medline: 18 Jun 1999
AB Obesity which is defined as accumulation of excess body fat, is a major cause of atherosclerotic vascular disease in industrial countries. Recent advances in the biology of adipose tissue have revealed that adipose tissue is not simply an energy storage organ but it also secretes a variety of molecules which affect the metabolism of the whole body. Through a systematic search of active genes in adipose tissue, we found that adipose tissue, especially visceral fat expressed numerous genes for secretory proteins (about 30% of total genes analyzed). Among them, plasminogen activator-1 (PAI-1), which is a regulator of the fibrinolytic system, was overexpressed in the visceral fat in an animal model of obesity. Plasma levels of PAI-1 were closely correlated with visceral fat adiposity. Thus, PAI-1 secreted from visceral fat may play some role in thrombotic vascular disease in visceral obesity. Adiponectin, a novel adipose-specific gene product, which has a matrix-like structure, is abundantly present in the bloodstream. Dysregulated secretion of adiponectin may be related to vascular disease in obesity. Biologically active molecules secreted from adipose tissue (adipocytokines) may have important roles in the development of atherosclerotic disease in obesity.